free from fever, but with more of the obscuration of the intellect, coming, at times at least, quite suddenly. Nothing further could be found to explain his condition except the state of the urine, with pus and blood, and obviously great pain in passing it. The cerebral symptoms, however, seemed again too distinct to be ignored, and I had once more to revise my diagnosis, saying that the patient had evidently meningitis, as at first stated. He died on October 15th.

Before the post-mortem examination, I stated to Dr. Joseph Coats, who made it, that I believed we had had both meningitis and enteric fever in the case; but I could not think of any proper explanation of the extreme emaciation, or of the urinary symptoms.

The examination showed the intestinal ulceration characteristic of enteric fever, but the ulcers were far advanced toward cicatrization; they were quite clearly *not* of the tubercular form. There was perfectly distinct and typical tubercular meningitis, and extremely advanced forms of tubercular disease in the kidneys and the bladder.

The tubercular disease of bladder and kidney at once explained both the wasted appearance and the urinary symptoms. The perfectly-clear character of the urine at my first visit had kept me from thinking of this; the disease was no doubt present there long before that, but probably underwent rapid development after the catheterization.

The combination of symptoms of meningitis with those of the fever was most perplexing. The meningitis seemed to be so far arrested in its progress by the development of the fever, and reasserted itself with its subsidence.

DISEASE OF THE MID-BRAIN REGION,

WITH SPECIAL REFERENCE TO OPHTHALMOPLEGIA, AND A NOTE ON POST-HEMIPLEGIC ATAXIA.[1]

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It is my intention in this paper to analyze as briefly as possible a number of cases in which oculomotor symptoms have played an important *rôle*. I have been fortunate in obtaining two autopsies which furnished good proof of the value of those symptoms commonly supposed to indicate disease of the mid-brain region, and several other cases that were subjected to careful clinical examination, will help to enforce the points which I wish to make.

It is only within recent years that the mid-brain region has acquired great clinical and practical value. This is due largely to the anatomical and pathological studies of Westphal (¹), Spitzka (²), Leube (³), and Wernicke (⁴), who have taught us to discriminate between the various subdivisions of the oculomotor nuclei, and have given us anatomical explanations of the many and complicated phenomena in polio-encephalitis superior (Wernicke), and in other conditions associated with ophthalmoplegia externa and interna.

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[1] Read at the meeting of the American Neurological Association, 1890.

Among other things, I wish to show that we should not be too ready to make the diagnosis of polio-encephalitis superior wherever and whenever partial ophthalmoplegia (external and internal) constitute the main symptoms of the affection. A diagnosis hastily made is often badly made.

CASE I.—My first case is one of unusually severe tubercular affection of the brain in a young child. The main points of the history, which I have been able to complete through the kindness of several colleagues, are these:

The child, E. S., aged three years, was first seen about the middle of November, 1889, by Dr. J. A. Meeks, in the Eye and Ear Department of the Northern Dispensary. At that time the little girl had double ptosis, but no other ocular paralysis was observed. The child was dull and listless. The pulse ranged between 165 and 160, but the temperature was normal. She was referred to the care of Dr. Seguin, at the Manhattan Eye and Ear Hospital, where Dr. Booth[1] took a very careful history. From his notes of December 9th I gather that the mother had noticed a change in the child's disposition since an attack of measles in the previous April. The child did not care to play with other children; preferred to sit quietly by herself all day long; staggered in walking, and occasionally fell. The child had no epileptic attacks, and had vomited but once. At this time (December, 1889) the child was stupid and deliberate in all her movements; walked with her feet widely apart; no staggering; very little strength in the hands. Right hand weaker than left. No anæsthesia or ataxia; knee-jerks absent. Paresis of both levatores palpebrarum, the pupils being half covered; no other paralysis of ocular muscles. No nystagmus. Pupils equal, moderately dilated, reacting to light and during accommodation. There was at this time a suspicion of optic neuritis. Pulse 140, and temperature of axilla 100.2°.

The child was brought to my department at the Polyclinic December 28, 1889. It was not known at the time that she had been seen by Drs. Meeks and Booth. A careful examination of her condition made before the students of the Polyclinic showed that there was double and almost complete ptosis. There was no upward or downward movement of either eye; both internal recti muscles were thrown into a clonic spastic condition when an attempt was made to use them. Both internal recti were capable of very slight movements, but all other external ocular muscles were completely paralyzed. Yet, strange to say, even at this late day the accommodation reflexes were distinct, and there was slight contractility to light in both pupils. A slight left facial paresis was noticeable. Vision was very much impaired. Although in a condition of semi-stupor, the child walked before the class, and exhibited in so doing most distinct staggering, which reminded one of typical cerebellar gait. She walked with a broad base, and showed distinct tendency to fall to the right side. The oculist (Dr. Webster) reported a distinct plaque of choroidal atrophy below the macula of the left eye. The reflexes were exaggerated, and there were very evident occipital headaches. The right hand was weaker than the left, but otherwise the use of all four extremities was entirely normal.

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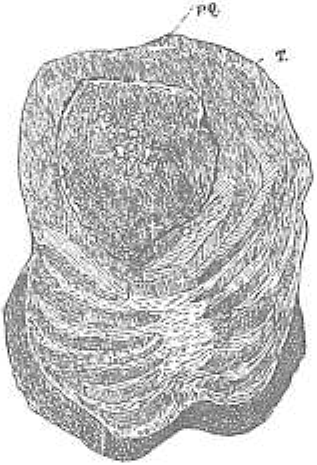
[1] I am indebted to Dr. Booth for his kindness in putting the hospital records at my disposal.

The diagnosis of tumor of the corpora quadrigemina was made, and this tumor was supposed to be associated with a general tubercular meningitis. While the ocular symptoms pointed to the region of the corpora quadrigemina, the subacute character of the onset, the involvement of the sixth and seventh nerves, the weakness of one upper extremity, and the occipital headaches, indicated a general and widespread cerebral affection. The tubercular character of the disease was diagnosticated from the general cachectic appearance of the child, from the peculiar development of the symptoms in the last weeks, and from the information given us, that an uncle of the child had just died of phthisis.

The child grew rapidly worse, and could not be brought to the clinic. At my request Dr. Peterson kindly visited the child at its home and kept track of it to the end. The child had occasional convulsive seizures, grew blind, became comatose, and two weeks before death developed left hemiplegia. There was no change in the eye symptoms, unless the light and accommodation reflexes disappeared, but this point could not be positively determined, owing to the comatose condition of the child. The child died February 4, 1890, four months after the onset of the symptoms.

By strategy and Dr. Peterson's persistence an autopsy was obtained, and the brain was removed by him. The dura was adherent to the skull, and had to be removed with the calvarium. The quantity of subdural fluid was slightly increased. There was discovered at once a tumor (solitary tubercle) near the right lateral sinus, about one centimetre in diameter and pressing upon the lateral edge of the cerebellum, producing thrombosis of the lateral sinus. Other tubercles surrounded by large areas of softened tissue were found in the cerebellum. An examination of the brain at my leisure showed that the hemispheres were healthy, with the exception of small tubercular deposits along the bloodvessels. The cerebellum was the seat of most profound changes, containing several large solitary tubercles. At the base there were several uncommon conditions; there was an unusual thickening of the pia, with small tubercular deposits between the corpora mammillaria and the optic chiasm, and in the interpeduncular space, the thickening here being so great, though probably of recent date, that both third nerves, instead of lying across the crura, after removal of the brain, pointed backward, and the right sixth was twisted out of its normal position. The brain was not cut into until it had hardened for about a week in a two-per-cent. solution of bichromate of potash. Cutting through the level of the corpora quadrigemina a tumor was found, as anticipated, the size of which will be best appreciated by an examination of Fig. 1. The brain was allowed to harden for many weeks in bichromate solutions of increasing strength, and later on in alcohol.

The sections were made from parts embedded in celloidin, and variously stained. It will be observed that there is no trace of the aqueduct of Sylvius in sections passed through the anterior quadrigeminal bodies, while in sections through the lower half of the pons one can observe the increased dilatation of the ventricle.[1] There is every reason to suppose, from a comparison of the symptoms and from the study of the anatomical specimen, that the tumor began to grow from the central gray substance. The tumor occupies almost the entire tegmental division of the crus, and has left only a very small portion of the corpora quadrigemina and the brachia intact.



Vertical section passing through the posterior quadrigeminal bodies.
T. Tumor. PQ. Posterior quadrigeminal bodies.

Reviewing the history of this case, and comparing the clinical symptoms with the results of the post-mortem examination, we are struck by the fact that the one solitary tubercle in the vicinity of the corpora quadrigemina produced more mischief than the large number of lesions elsewhere in the brain. The occipital character of the headache, and the cerebellar staggering, are the only symptoms which can be ascribed to the tubercles in the cerebellum, though both these symptoms might be due to disease of the quadrigeminal region. Indeed, Nothnagel (5) has recently claimed that cerebellar walk and nuclear affections of the oculomotor nerves are the most distinct symptoms pointing to disease of the corpora quadrigemina. In this he has been supported by the few authors who have reported cases since the publication of Nothnagel's article, notably by Alfred Christ (⁶) in an able paper on this subject.

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[1] Further anatomical details are omitted.

If we consider the intimate anatomical relation between the corpora quadrigemina and the cerebellum through the anterior peduncles of the cerebellum, there is nothing surprising in the fact that cerebellar staggering should be a symptom common to disease in both of these regions. In this case it will be impossible to determine whether this one symptom was due to disease of the corpora quadrigemina or to the involvement of the cerebellum; but, inasmuch as the ocular symptoms preceded all other symptoms for some considerable length of time, it is safe to say that cerebellar staggering is not one of the *early* symptoms of disease of the quadrigeminal bodies, though it is undoubtedly a symptom that is developed in the later stages of such disease. An examination of sections through the pons showed that the sixth and seventh nerve nuclei were entirely normal; they can, therefore, not be held responsible for symptoms pointing to involvement of these nerves, but this must unquestionably be placed to the account of the basilar meningitis.

Certain it is, that this meningitis was not the cause of the oculomotor symptoms; for months, or at least for weeks, the symptoms were distinctly nuclear; both levators were affected long in advance of other muscles, and the internal recti muscles retained some function long after the other muscles supplied by the oculomotor nerves had been completely paralyzed. In spite of the manifold morbid conditions, it is most remarkable of all that the ciliary muscles and the sphincter iridis should have remained exempt during the entire period of observation—a striking proof of the fact that the nuclei of these two muscles are at some distance from those governing the other and external muscles. Moreover, the fact that the levatores palpebrarum were affected to the exclusion of other muscles for a long time would point to the main cell-nests of the third nerve nuclei as the original seat of the tumor. Whether the tumor invaded both sides at once, or whether the decussating fibres from the main cell-nests are responsible for the bilateral character of the nuclear symptoms cannot be determined by cases of tumor.

The chief inference to be drawn from this case is that symptoms of ophthalmoplegia externa and interna, if associated with symptoms pointing to a new-growth within the brain, will serve to locate that growth in the vicinity of the corpora quadrigemina.

Considering the compactness of all cerebral structures in the mid-brain region, one would expect that there could not for an instant be any difficulty in making a differential diagnosis between cases of tumor of this region and a chronic inflammatory process. One would expect a far greater number of pressure-symptoms in the former than in the latter instance, yet the following case will serve to illustrate this difficulty:

CASE II.—In August, 1889, I was asked by the late Dr. Cocks to see Mrs. L., aged forty years. She had been married for many years, but had never been pregnant; was always in excellent health until five years ago, when she began to be troubled with headaches and vomiting, cerebral in character, and a peculiar sensation in the head on looking upward. At the same time she noticed that she saw things double, particularly on the street and on looking through an opera-glass. These symptoms had continued, with slight intermissions, up to the day of examination, except that the paresis of the eyes had very much increased. She was in the habit of keeping the left eye closed altogether, in order to have single vision. Had vomiting-spells occasionally, and some few fainting-spells many years ago, which may have been due to profound anæmia. My examination was entirely negative, except as regards the eyes. There was marked paresis of both internal recti in united and single action; external recti act fairly well, but nystagmus movements result if the attempt is made to bring the eye to the outer canthus. Upward movement of the eyes barely possible; downward movement also limited. Pupils react normally to light and during accommodation. Ptosis is said to have existed, but is not present now. Knee-jerks normal, and sensation absolutely so. At this time I made the diagnosis ophthalmoplegia nuclearis externa, probably due to a polio-encephalitis chronica, but remembering the occasional vomiting, I mentioned the possibility of tumor; not really believing it, however, but yet feeling disposed to give the patient the benefit of the doubt, I urged the continuance of active mixed treatment.

The further course of events was rather surprising. About two months after I had seen the patient for the first time, I had the privilege of examining her again. She was troubled severely with headaches and vertigo, and had been alternately drowsy and wakeful. In addition to these symptoms, she developed incoördination and characteristic cerebellar staggering. On October 5th, Dr Cocks reported to me that he suspected optic neuritis; and on October 17th there was very marked double optic neuritis, and the patient was no longer able to stand without support. The symptoms of tumor were so distinct that the idea of a polio-encephalitis superior had to be abandoned. The patient died October 31st, having become almost blind and wholly unable to leave her bed for some days before death. In spite of the most strenuous efforts, the husband refused an autopsy.

The idea of tumor in this case can scarcely be rejected, yet no one, I think, would have supposed that for more than five years the growth of the tumor in this region would be so gradual as to affect certain cell-nests only, and not the entire nuclei; for it will be remembered that the sphincter and ciliary muscles were entirely free as long as a detailed examination could be made. If asked to carry the diagnosis still further, it would be fair to say that there may have been a gummatous infiltration of this region, as has been found in other cases, notably in one of Thomsen's (¹); that this increased very slowly, diminishing at times and again growing anew, thus bringing about a chronic and variable group of symptoms. This inconstancy[1] of symptoms in specific disease of the cerebral nervous system has been correctly insisted upon by Dr. Oppenheim (⁶), of Berlin, in a very able discussion of the subject. In the case I have just discussed there is still another possibility—there may have been a mere gummatous infiltration of the oculomotor nerves which caused symptoms resembling those of nuclear disease, and toward the latter part of the disease the growth of a large gumma may have given rise to those symptoms which pointed so clearly to the development of a neoplasm.

But whether we had here to deal with gumma or mere gummatous infiltration, it is important to note that the manner in which the case terminated disproved the diagnosis of polio-encephalitis which for months, if not years, was the only safe diagnosis to make.

In this connection I am reminded of the difficulty of diagnosis in still another respect: since our anatomical knowledge has been worked out to the finest details, it is a point of some practical value to diagnosticate the character of the morbid process. Syphilis—ocular paralyses, gummatous infiltration of the base—is the natural sequence of our thoughts in many cases, and yet the symptoms which we observe frequently make it difficult to determine whether the disease is of nuclear origin, or whether the oculomotor root-fibres are affected by specific disease of the base of the brain. The following case will illustrate this point:

CASE III.—During many months, Dr. Gruening, who was kind enough to refer the patient to me, and myself, have had repeated occasions to examine a young gentleman from the western part of New York State. The patient is now thirty-three years of age, single; his family doctor reports that he had chancre with secondary eruption fourteen years ago, was thoroughly treated, no trace of syphilis since that time. Has never had headaches, and extremely well otherwise. Has been a hard worker for years. A year ago returned from an extended tour abroad. During this tour had spent weeks on the Lybian desert, and is inclined to think that the glaring white sand (he never wore glasses) is the cause of his trouble. Shortly after his return he noticed that he began to see double and that his eyes drooped. My examination of the ocular condition brought out the following facts: Left ptosis covering three-fourths of pupil; eyelid can be raised a little by great effort. Reflexes to light and accommodation almost normal. Left pupil possibly a little sluggish to light. Left abducens completely paralyzed. All other movements of eyes are perfect, inward, upward, and downward. Dr. Gruening found swelling and redness of the left optic disk. The patient was placed upon a most vigorous course of the iodides, taking as much as one hundred and fifty drops of a saturated solution of the iodide of sodium three times a day for weeks. This he bore extremely well, his general health remaining excellent. His condition since has varied somewhat, the rectus externus of the right eye showing slight paresis at times, and then again disappearing.

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[1] And yet this statement should be accepted with some reserve, for in the case of glio-sarcoma reported by Christ (loc. cit.) the symptoms were variable enough.

After months of treatment, and several months during which treatment was entirely suspended, the condition has changed in some respects. The left eye is now altogether better than the right eye. The left ptosis has been considerably relieved. The left rectus externus brings the eye to within one-eighth of an inch of the canthus, and all the other ocular muscles have normal function, but now the right eye is worse. The right rectus externus can bring the eye to within one-fourth inch only of the canthus. Upward and downward movements are normal, but the right pupil responds very sluggishly to light, while it does fairly well during accommodation. Right ptosis is more marked than left.[1]

In this case it is worth noting that an actual improvement of the condition of the left eye set in some time after all anti-syphilitic treatment had been abandoned, and that, while the left eye continued to improve, symptoms set in in the right eye very like those of the other eye, which had been the starting-point of the whole trouble. The entire history of the case is in favor of the specific origin of this trouble, yet if we remember that there was ptosis of one eye, paresis of the rectus internus of the same eye, and involvement of the rectus externus, while the other muscles supplied by the oculomotor nerve remained intact, we see that we have a group of symptoms closely resembling those of nuclear disease. Yet I cannot conceive, in view of the marked improvement of the left eye, that such is the case, and we are therefore forced to the conclusion that a specific infiltration of the oculomotor nerves may simulate disease of the oculomotor nuclei. This would be a point of some importance in the differential diagnosis of those cases in which apparently nuclear symptoms occurred in individuals who give no history of previous specific disease.

Fournier(9), Rumpf(10), and Oppenheim(8) are all agreed as to the occurrence of partial ophthalmoplegia in cases of syphilitic disease at the base of the brain, and Thomsen's case shows that a nuclear paralysis can be simulated by a gummatous infiltration of the root-fibres.

I cannot help thinking that just as the larger peripheral nerves are mixed nerves, giving rise, under certain conditions of disease, to motor or sensory symptoms, and not necessarily to both, so the oculomotor nerve is a mixed nerve, and that certain diseases affecting the third nerve show a predilection for definite sets of fibres. The anatomy of the third nerve rootlets has not been so clearly studied that we can point out the fibres or sets of fibres going to each muscle of the eye; but there is already good reason to suppose that the innermost rootlets are those which represent the internal rectus. A comparison of this case with others leads me to believe that, while we should keep the possibility of nuclear lesion in mind in such cases as the one just described, a preceding history of specific infection, and the inconstant character of the symptoms, indicate specific disease of the third nerve roots, rather than of the third nerve nuclei.[1]

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[1] In Feb. 1891, the patient presented himself again. The ptosis of both eyes has disappeared entirely: the right eye is entirely well, and in the left eye there is nothing but a slight paresis of the rectus externus.

To the preceding cases we may add, properly enough, another in which the oculomotor symptoms were distinct, yet not so striking a feature as the peculiar disturbances of motion which lent a special interest to the case. The situation of the lesion in this instance is of special importance, by way of throwing some light upon the origin of post-hemiplegic disturbances of motion.

CASE IV.—The patient was a married woman, fifty-seven years of age at the time of death, December 2, 1889. Very nearly seven years previously patient felt dizzy one morning and found her vision rather blurred. This condition passed away, but returned again after fifteen minutes. She remained seated in her chair, was not unconscious for an instant, and had no trouble whatever in speaking. She attempted to walk, found this rather difficult, but did not fall. The difficulty increased, and by the following morning she had almost complete left hemiplegia. She could not open either eye. The attending physician noticed, on raising the eyelid, that vision was dim. What the position of the eyes was at this time could not be ascertained. Speech was also heavy and indistinct, but from this the patient recovered within three weeks. There was no defect of any other special sense. The hemiplegia was never recovered from. Patient became somewhat unruly and demented, and was finally taken to the Montefiore Home, where she was an inmate for many years. As far as could be determined, patient had never had syphilis, but there was very marked atheroma of all peripheral arteries: the patient was prematurely old. In addition to her left hemiplegia, she also had lost the use of her right leg, which had been amputated above the ankle on account of necrosis of the tibia fully six years ago. Being bedridden, she was covered with a succession of sores, and was altogether a revolting sight. The right eye at the time of my first examination was completely closed, the globe was rotated so that the pupil stood in the extreme outer corner of the orbital cavity; there was not the least possibility of movement inward, upward or downward. The pupil was widely dilated; no reflex to light; accommodation reflex, of course, could not be tested. There was rigidity of the left leg, and increased knee-jerks on both sides. The wrist-reflex was decidedly increased on the paralyzed side, but this left upper extremity was subject to the wildest ataxic movements. I call them ataxic, for they remind me of the extreme ataxic movements of the legs in the last stages of tabes dorsalis. If our patient was asked to use the paralyzed hand, say, in order to squeeze the physician's hand, this paretic extremity was whirled about so violently as to be a source of some danger to those near the bed. After a little, the arm would fall down from exhaustion, and remain quiet until aroused again. The patient's condition grew worse during the latter half of the year; she became extremely emotional and demented, took very little nourishment, and finally died December 2d. She was not intelligent enough during the time I observed her to enable me to make satisfactory sensory tests. The diagnosis of crus lesion of right side, probably softening from thrombosis, was corroborated by the autopsy.

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[1] This conclusion is supported by other cases to which I shall refer in a later article.

The autopsy was made a few hours after death. The dura was found adherent to the skull; the pia could be readily removed from the entire surface of both hemispheres. Nothing abnormal about the appearance of the convexity. An inspection of the base of the brain revealed at once the small size and extreme softness of the right crus as compared with the left. The right oculomotor nerve was as thin as a thread, the left about of normal size. The vessels of the brain were carefully examined, and no changes were found except in the post-cerebral artery of the right side, which was plugged by a firm thrombus that had evidently been formed *in situ*. Beyond the thrombus the artery had been reduced to a mere thread. The brain was handled after Meynert's fashion, the ganglia being dissected away from the hemispheres. The cortex was found entirely normal, and in all the organs above the crus there was nothing of any significance. The brain was hardened in the usual way in Müller's fluid and alcohol. Every part except that which was the seat of the lesion hardened well. After the brain had become slightly hard, the crura were cut across, showing distinctly the absolute disintegration and atrophy of the right crus, the process having exceeded the middle line by a fraction of an inch. Sections were laid throughout the pons, medulla, and spinal cord; and, almost at every level, distinct degenerated fibres were found in the pyramidal tract and in both lateral tracts, yet the degeneration was not as pronounced as one might have expected from disease of such long standing. Sections of the right oculomotor nerve exhibited complete degeneration of the fibres, while the left third nerve also showed some degeneration on cross-section, though there were but very few fibres thus affected.

The occurrence of ataxic movements in cases of crus lesion is the one point to which I wish to call attention, while the ocular condition will serve to show how radically different the symptoms are in cases in which the entire nerve is affected by disease. The cortex and the sub-cortical ganglia were entirely normal, yet this peculiar disturbance of motion was like those we are accustomed to see in the wake of infantile cerebral palsies, a full description of which has been given in a previous paper by Dr. Peterson and myself (11). In children the lesion is very often cortical, occasionally it is in the vicinity of the ganglia, but in the case with which we are at present concerned the same order of symptoms is produced by a lesion much further caudad. I accept this case as proof of the correctness of the views previously expressed, that these peculiar movements may be due to the removal of inhibition, but that there must be some irradiation of nerve force from one half of the central nervous system to the other, and that this irradiation takes place at any diseased level as low down as the pons and medulla, and possibly even in the spinal cord. Associated movements are explained readily enough on this hypothesis, but why these extreme and violent ataxic movements should occur by transmission of motor impulses through such an imperfect motor tract as we had before us is not so easy to see.

It would seem as though the motor impulses in this case had been transmitted from the healthy side—a sort of overflow of volitional energy from the sound side which is not restrained by the ordinary inhibitory force; the removal of this inhibitory force being due to the lesion in question. From all of which we make the very natural deduction that the two halves of the central nervous system in health coöperate by antagonizing each other's forces, each half in health placing a check upon the superabundant energy of the other.

The preceding communication will serve to illustrate the following points:

1. The slow onset of oculomotor symptoms associated with ataxia and vomiting point to a neoplasm in the vicinity of the corpora quadrigemina.
2. The diagnosis of polio-encephalitis superior (Wernicke) should not be made unless tumor of the quadrigeminal region can be positively excluded.
3. Paralyses of one or more muscles, and not of all muscles supplied by the oculomotor nerve, do not necessarily imply a nuclear lesion. This is particularly true of syphilitic cases, in which an affection of the nerve rootlets may give rise to symptoms exactly like those of nuclear disease.
4. Post-hemiplegic ataxic movements may result from lesion of the crus, and probably from lesion in any part of the motor tract.

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THE TREATMENT OF PENETRATING GUNSHOT WOUNDS OF THE ABDOMEN,

WITH AN ANALYSIS OF ONE HUNDRED AND SIXTY-FIVE CASES TREATED BY LAPAROTOMY.

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IN the *Revue de Chirurgie* of February, 1890, in an elaborate article fifty-three pages in length, two well-known French surgeons, M. Reclus